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Adaptation of cancer cells from different entities to the MDM2 inhibitor nutlin-3 results in the emergence of p53-mutated multi-drug-resistant cancer cells

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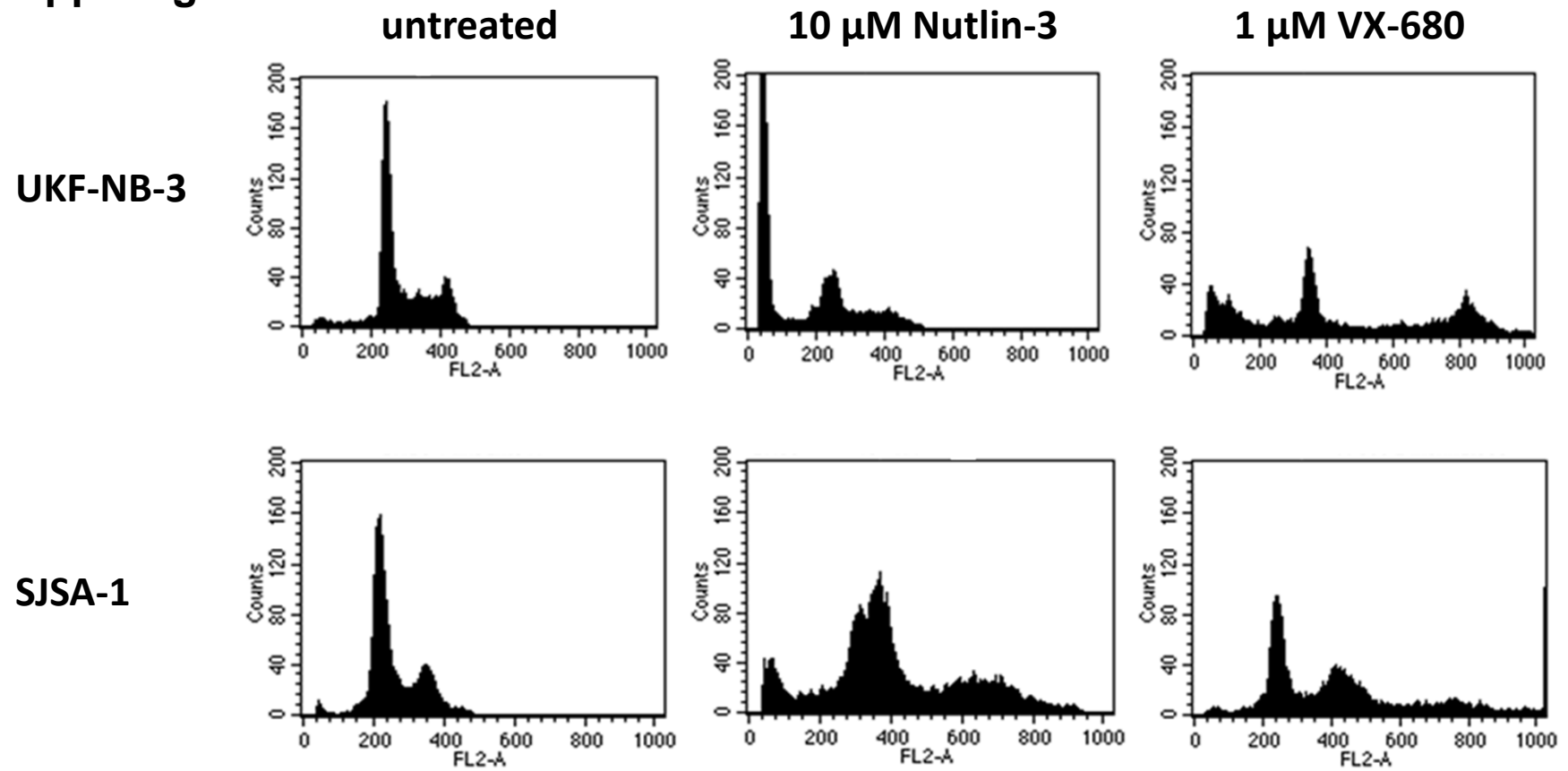
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Suppl. Figure 1



Suppl. Figure 1. Detection of DNA content in different cell lines after treatment with nutlin-3 or the aurora kinase inhibitor VX680. Cells were fixed with 70 % ethanol for two hours at -20°C. The cellular DNA was stained using propidium iodide (20 µg/ml, purchased from Sigma-Aldrich Chemie GmbH, Munich, Germany) and analysed by flow cytometry (FacsCalibur, BD Biosciences, Heidelberg, Germany).

Previous investigations had shown that transient treatment of osteosarcoma cells with nutlin-3 resulted in the formation of tetraploid clones (Shen et al., 2008). Here, nutlin-3 induced tetraploidy in osteosarcoma SJSA-1 cells but not in UKF-NB-3 cells. In contrast, treatment with the aurora kinase inhibitor VX-680 that is known to induce tetraploidy (Harrington et al., 2004) resulted in a fraction of cells with a DNA content $\geq 4N$.

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